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Case Report

Propriospinal myoclonus following spinal anesthesia for knee arthroscopy

Myoclonus due to spinal anesthesia

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Abstract

Spinal myoclonus (SM) after neuraxial anesthesia is a rare but distressing complication for anesthesiologist. Propriospinal myoclonus, a variant of spinal myoclonus, is even rarer and is a disorder characterized by sudden, shock-like, involuntary jerks originating from axial muscles.

We present a case of a patient who developed propriospinal myoclonus 135 minutes after spinal anesthesia.

Arthroscopy under spinal anaesthesia was planned for a patient with meniscopathy. Spinal anaesthesia was performed with 10 mg hyperbaric bupivacaine and 100 mcg morphine. Myoclonic movements in the upper extremities and trunk of the patient started 135 minutes after spinal anaesthesia. Diazepam 10 mg slow bolus followed by 10 mg/30 min infusion was administered and the frequency of myoclonic movements decreased but did not completely disappear. Diazepam infusion was repeated and myoclonic movements disappeared approximately 6 hours after spinal anaesthesia.

In conclusion, anesthesiologists should be aware that this rare condition is a temporary complication with no sequelae.

Keywords

Arthroscopy, Myoclonus, Propriospinal Myoclonus, Spinal Anesthesia

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Introduction

Spinal anesthesia is very common in daily anesthesia practice, due to its advantage such as protection from airway complications. Anaesthesiologist may encounter rare but serious neurological complications of spinal anaesthesia. One of these complications is Spinal Myoklonus following Neuroaxiel Anesthesia (SM-NA) and only 23 cases have been reported in the literature so far. SM-NA is a movement disorder originating in the spinal cord and causes transient myoclonic involuntary movements of the extremities or trunk [1].

Spinal myoklonus is of two types, segmental and propriospinal myoklonus. Propriospinal myoclonus is a rare disorder characterized by sudden, shock-like, involuntary jerks that arise from the axial muscles and spread both rostrally and caudally to other myotomes through slow polysynaptic pathways. It can be secondary to spinal cord lesions; additionally, it can develop as an adverse effect to the administration of several drugs, including neuraxial local anesthetics [2].

We aimed to present a patient who underwent spinal anaesthesia for meniscopathy surgery and developed SM-NA. The aim of this case report is to enable anaesthesiologists to recognise SM-NA, which is a rare complication, and to draw attention to the fact that benzodiazepines can be used to suppress symptoms, although they do not provide a complete cure. Informed consent was obtained from the patient.

Case Report

A 34-year-old, 175 cm, 97 kg male patient was diagnosed with meniscopathy and arthroscopy was planned. The patient had a history of inguinal hernia operation under spinal anesthesia 1 year ago, thyroidectomy operation 6 years ago and levothyronine use. Preoperative laboratory values were within normal limits. Standart monitoring (ECG, SpO2, non-invasive blood pressure) was performed. After antisepsis in the sitting position, 10 mg hyperbaric bupivacaine and 100 mcg morphine were administered intrathecally. After intrathecal injection, the sensory block level was evaluated as T6 dermatome and the patient was delivered to surgery. The patient's vital signs were stable during surgery and no additional medication was administered. After the 135-minute surgical procedure was completed, myoclonic movements started in the patient's upper extremities and trunk. At this time, the patient was conscious and cooperative. Blood gas and biochemical tests were repeated and no abnormality was found in the new results. The patient was given 2 mg midazolam two times. Myoclonic movements started to be seen in the lower extremities approximately 1 hour later. Neurology was consulted and neurologist recommended diazepam 10 mg slow bolus followed by diazepam 10 mg/30 min infusion and ordered Electroencephalography (EEG) and brain magnetic resonance imaging (MRI) to investigate the etiology. The frequency of myoclonic movements decreased considerably after diazepam. The patient, who was always conscious during this period, was transferred to the Intensive Care Unit (ICU). 10 mg diazepam was infused again in 1 hour and the myoclonic movements stopped completely in ICU. The patient was checked daily until discharge and no abnormal neurologic findings were observed. The patient was followed up by neurology and epileptiform changes were observed in the EEG taken 1 week later. 2 months later, MRI revealed that "the temporal horns of the lateral ventricles were more prominent on the left side in the coronal oblique sections taken from the hippocampus and temporal lobe. The volume of the hippocampus was slightly decreased, more prominent on the left". The neurologist stated that this structural abnormality was not a factor in the development of myoclonus. The patient who had no neurologic symptoms in the last 3 months was not started any drug treatment by the neurologist.

Discussion

Myoclonus is the involuntary twitching of muscles that is caused by brief electromyographic discharges. Myoclonus can arise from either the central or peripheral nervous system. Central nervous system myoclonus can originate from cortical, subcortical, brainstem and spinal cord. SM is a type of myoclonus originating from the spinal cord [3, 4] and is a very rare complication of spinal anesthesia.

The pathophysiology of SM-NA is not fully understood because there is no research showing that local anesthetics act on spinal neurons to produce myoclonus. Based on the reported cases, the most well-established characteristics of SM-NA are that it occurs frequently with hyperbaric local anesthetics, is more common in women, and its occurrence is unpredictable [1].

SM is classified into two types: segmental and propriospinal. Segmental SM typically originates within a few or several adjacent spinal segments, whereas propriospinal SM refers to myoclonic involuntary movements involving muscles innervated by many different segments. Propriospinal SM is extremely rare than segmental SM [5]. The propriospinal route is a pathway for transmitting stimulation among multiple spinal segments. Abnormal impulses originating from the lumbar spinal cord can ascend and spread upward through this pathway, resulting in myoclonic involuntary movements of the upper extremities and trunk [3, 4]. In our case, myoclonic movements were observed both in the extremities and trunk, so we think it is propriospinal myoclonus. Myoclonus in the trunk (17.4%) is more rare than myoclonus in the extremities (30.4%) [1].

Shiratori et al. reported 23 case reports in their literature review. 16 of these patients were female, suggesting that the incidence of SM-NA differs according to gender. SM-NA has been observed at younger ages in females and at older ages in males [1]. A feature that makes our case different is that our patient was a young male. This may be interpreted that the incidence of SM-NA differs according to age as well as gender. In most of the cases, SM-NA started within the first 3 hours after spinal anesthesia and usually ended within 12 hours. In our case, it started within 3 hours and ended after 6 hours. In 10 cases, recovery occurred spontaneously, while in 13 cases anticonvulsant drugs were empirically administered [1]. In the drug-treated cases, it is not known whether the drug completely cured SM-NA, because the clinical picture improves spontaneously with the resolution of the effect of the local anesthetic. Nevertheless, it has been reported that benzodiazepines may be effective or semi-effective in the rapid suppression of SM-NA. At first, our patient was tried to be treated with midazolam but no response was obtained. On the recommendation of neurology, diazepam 10 mg slow

bolus followed by infusion (10 mg/30 min) was started and the frequency of myoclonus decreased but did not stop completely. After taking diazepam again at a rate of 10 mg/hour, the myoclonus stopped. Meanwhile, approximately 6 hours had passed since spinal anesthesia. The cure achieved after 6 hours may also be related to the dissolution of the effect of the local anesthetic.

It has been reported that no abnormal findings were found in patients' EEG in the middle of the SM-NA event and after the SM-NA passed [2,6]. In our case, EEG could not be taken at the time of the event but generalized epileptiform changes were observed in the EEG taken 1 h later.

The coexistence of reduced activity in the suprasegmental descending inhibitory pathway, increased excitability of inhibitory interneurons, and the abnormal hyperexcitation produced by the recovery of alpha motor neurons in the anterior horn are the mechanisms of SM-NA generation. When diagnosising of SM-NA, the following conditions causing myoclonus may need to be excluded; myoclonus due to neuraxial opioid high dose therapy and drug-induced dystonic movements. Neuraxial opioids can also cause myoclonus as a side effect, but this side effect is more likely to be seen in long-term or high-dose use [7]. In our case, intrathecal morphine was used, but the dose used (100 mcg) was not high and it was a one-time use.

SM-NA can also be confused with drug-induced dystonic movements. Movements in SM-NA are faster than in dystonia and are lightning-like muscle jerks that usually develop in the extremities. Dystonic movements are characterized by sustained muscle contractions that cause abnormal twisting-like movements and postures involving the trunk and limbs, which can also affect the facial muscles. In our patient, there was no abnormal posture and the facial muscles were not affected. The following drugs have been blamed for the etiology of drug-induced dystonic movements; antihistamines, antimalarials, antiseizure medications, calcium channel blockers, cholinergic agonists, lithium, methylphenidate, metoclopramide, selective serotonin reuptake inhibitors, tricyclic antidepressants [8]. But none of these drugs were used in our patient.

Six of the cases reported as SMNA had previously undergone surgery under spinal anesthesia and did not develop myoclonus. Our patient also underwent inguinal hernie operation under spinal anesthesia 1 year ago and did not experience any complications to spinal anesthesia. The occurrence of SM-NA is unpredictable because there are patients diagnosed with SM-NA who did not have any problems in previous spinal anesthesia experiences, and there are also cases where SM-NA did not recur or recurred in subsequent spinal anesthesia applications. It has been reported that a history of SM-NA is not a contraindication for re-NA, but SM-NA recurrence should occur within 1 year after the first episode. When patients who have undergone SM-NA within the last 1 year undergo a new operation, it is recommended to be prepared for possible SM-NA or to choose an anesthesia method other than neuraxial anesthesia [1].

Conclusion

Spinal myoclonus is a very rare, unusual event that can occur after administration of spinal anesthesia. It is a self-limiting adverse event and usually resolves after the drug wears off.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or compareable ethical standards.

Conflict of interest

The authors declare that there is no conflict of interest.

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